> d his

(FILE 'HOME' ENTERED AT 12:29:05 ON 15 SEP 2003)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT' ENTERED AT 12:29:28 ON 15 SEP 2003

L1 10 S (LDL RECEPTOR) AND (GAMMA SECRETASE)
L2 4 DUPLICATE REMOVE L1 (6 DUPLICATES REMOVED)

=>

LVCook
9/15/03

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=> d his

     (FILE 'HOME' ENTERED AT 11:32:37 ON 15 SEP 2003)

     FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT' ENTERED AT 11:33:02 ON
     15 SEP 2003
L1             1 S (LDL RECEPTOR TRANSMEMBRANE DOMAIN)
L2           221 S (LDL RECEPTOR) AND TRANSMEMBRANE
L3             3 S L2 AND (TERMINAL TAIL)
L4             8 S (LDL RECEPTOR) AND (TERMINAL TAIL)
L5             4 DUPLICATE REMOVE L4 (4 DUPLICATES REMOVED)

=>
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L1 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1999:28420 BIOSIS
DN PREV199900028420
TI Promoter structure and transcriptional activation with chromatin templates assembled in vitro: A single Gal4-**VP16** dimer binds to chromatin or to DNA with comparable affinity.
AU Pazin, Michael J.; Hermann, Jason W.; Kadonaga, James T. (1)
CS (1) Dep. Biol., 0347 Pacific Hall, Room 2212B, Univ. California San Diego, 9500 Gilman Dr., La Jolla, CA 92093-0347 USA
SO Journal of Biological Chemistry, (Dec. 18, 1998) Vol. 273, No. 51, pp. 34653-34660.
ISSN: 0021-9258.
DT Article
LA English
AB To gain a better understanding of the role of chromatin in the regulation of transcription by RNA polymerase II, we examined the relation between promoter structure and the ability of Gal4-**VP16** to function with chromatin templates assembled in vitro. First, to investigate whether there are synergistic interactions among multiple bound factors, we studied promoter constructions containing one or five Gal4 sites and found that a single recognition site is sufficient for Gal4-**VP16** to bind to chromatin, to induce nucleosome rearrangement, and to activate transcription. Notably, we observed that Gal4-**VP16** binds to a single site in chromatin with affinity comparable with that which it binds to naked DNA, even in the absence of ATP-dependent nucleosome remodeling activity. Second, to explore the relation between translational nucleosome positioning and transcriptional activation, we analyzed a series of promoter constructions in which nucleosomes were positioned by Gal4-**VP16** at different locations relative to the RNA start site. These experiments revealed that the positioning of a nucleosome over the RNA start site is not an absolute barrier to transcriptional activation. Third, to determine the contribution of core promoter elements to transcriptional activation with chromatin templates, we tested the ability of Gal4-**VP16** to activate transcription with TATA box- versus DPE-driven core promoters and found that the TATA box is not required to achieve transcriptional activation by Gal4-**VP16** with chromatin templates. These results suggest that a single protomer of a strong activator is able to bind to chromatin, to induce nucleosome remodeling, and to activate transcription in conjunction with a broad range of chromatin structures and core promoter elements.
CC Genetics and Cytogenetics - General *03502
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines *10062
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Replication, Transcription, Translation *10300
IT Major Concepts
Molecular Genetics (Biochemistry and Molecular Biophysics)
IT Chemicals & Biochemicals
chromatin: Gal-4-**VP16** dimer binding affinity, in vitro template assembly; DNA: Gal-4-**VP16** dimer binding affinity; **Gal14-VP16** promoter: structure
IT Miscellaneous Descriptors
transcriptional activation

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